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ORIGINAL ARTICLES.

THE DANGER OF THE SPONTANEOUS CURE OF SENILE CATARACT.

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OMAHA, NEB.

THAT a spontaneous cure of senile cataract sometimes occurs is well enough known. I have seen four such cases, and it is highly probable that if cataract patients lived longer and could keep out of the hands of the oculists, a large proportion of them would be more or less completely relieved in this way. But unfortunately for Nature's statistics, I have to report that in three of my four cases the eye has been lost by glaucoma.

The case in which sight was retained had nothing unusual about it for such a case. In a German, 66 years of age, a cataractous lens became partially absorbed, probably through liquefaction of the cortex and rupture of the capsule, and the nucleus sank down, so that when I first saw him the upper third of the pupil was free and the vision, with the refraction corrected, was $^{20}/_{LXX}$. The other three cases were as follows:

CASE 1.—A. F., German, 63 years of age, came to me March 15, 1887. R. E. normal except for slight peripheral opacities in the lens. V.=$^{20}/_{XX}$—. L. E. cataract two-thirds mature. V.= fingers at three feet; light sense and projection normal. A preliminary iridectomy was made in the left eye, and he was told to return when the sight of the right eye became so poor as to interfere with his work. I heard nothing from him for five years, when he returned, stating that for some months the left eye had been entirely blind and quite painful. I found the right eye practically unchanged. Vision was $^{20}/_{XXX}$, and the cataract showed very little sign of progress. In the left eye nothing remained of the cataract but a small brown, freely movable, nucleus behind the lower part of the iris, The upper part of the pupil was entirely clear and permitted a good view of the fundus. The eye was very hard, painful and absolutely blind. I find no note as to whether the nerve was excavated or not, but I think it was. I extracted the nucleus from the left eye whereupon it became softer and free from pain. I have heard from him recently and he still has good sight in the right eye.

CASE 2.—Mrs. P., 65 years of age, came to me March 2, 1896, with a nearly mature cataract in the left eye; the lens had been extracted from the right eye ten years ago. R. E. V., with correction, =$^{20}/_{XL}$, L. E. V.=fingers at six feet. She was told to return in a month or so, but although she lived but a short distance from Omaha I did not see her again for two years, and when she returned I found that the opaque cortex of the left lens had been absorbed and the nucleus had sunk down so as to leave the upper third of the pupil free. A red reflex could be obtained from the fundus but no details could be seen. The tension was $+1^1/_2$ and the eye was absolutely blind. The other eye still had vision =$^{20}/_{L}$, and showed no signs of glaucoma. I have heard from her within a few days and she reports that she still sees fairly well with the right eye.

CASE 3.—In this case the patient was not seen until the process had reached its final stage. The patient, an Italian, about 70 years of age, had had the left lens extracted with an iridectomy several years previously with a fair result, and the eye remained good, although I did not test its vision

accurately. After the operation on the left eye the right lens had become opaque, but he had not sought an operation as the sight of the left eye sufficed. During the last six months the right eye had been painful at intervals and he came to me suffering severely. I found the eye to be deeply congested, very hard and entirely blind. The cornea was so hazy that I could not determine the exact conditions in the anterior chamber, but as it was evident that the lens was opaque, I decided to extract it to relieve the pain. On operating I found that the cortex had all been absorbed and nothing was left of the lens but a very small nucleus which had found its way through the pupil into the anterior chamber. The man was lost sight of soon after the operation.*

The frequency with which a spontaneous cure of cataract is associated with glaucoma has been previously noticed, particularly by Mitvalsky (*Centralblatt f. Augenheilkunde*, October, 1892, p. 297), and Reuss (*Ibid.*, February, 1900, p. 39), but they seem inclined to regard the glaucoma as a

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*NOTE.—Since writing the foregoing I have seen a fourth case of glaucoma from or with the spontaneous cure of cataract: A man, 77 years of age, was seen by me October 15, 1900. He had a cataract removed three years ago from the right eye; the left eye became blind from detachment of the retina (Dr. Webster) eighteen years ago; this eye was never operated on. Relatives state that the pupil of the left eye became as white as did the right eye some years ago, but that for three or four years the pupil has been black again.

Status præcens: Right eye, aphakial, pupil round; upper half of the retina detached. V.= fingers at six to eight feet; tension normal. Left eye, T.+1; iris pressed slightly forward; pupil round and slightly dilated; fundus lined with grayish-brown membrane, in folds, but slightly movable; very few vessels to be seen; vitreous clear; no sign of lens or capsule behind the pupil, but when the patient looks down the grayish-white lens, apparently of about two thirds the normal diameter, can be plainly seen, lying far forward on the floor of the vitreous chamber, the anterior edge tilted up against the ciliary processes or the root of the iris. V.=0.

cause rather than an effect of the spontaneous cure of the cataract. It may be that they are right and that I have been unduly impressed by having more than my share of such cases. To establish with any certainty the secondary nature of the glaucoma in my cases I ought to be able to give a history of improved vision after the development of the cataract and before the appearance of the glaucoma, and this I can not do. One of the patients had an indistinct impression of something of the kind but her testimony was too indefinite to be used; but when we consider the conditions attending the spontaneous cure of cataract it is easy to conceive how the first steps in the process might cause glaucoma before enough change had occurred in the sight to be noticed by a patient with one good eye.

Nature's cures generally take place either through the spontaneous dislocation of the entire lens downward or more frequently through the absorption of the liquefied portion of a Morgagnian cataract, either on account of a rupture in the capsule or, possibly, without such a rupture. In any case the lens, or what is left of the nucleus, is in a position to excite ciliary congestion and perhaps glaucoma by bouncing about upon the ciliary process, or perhaps by direct pressure upon the outlet of the anterior chamber, either from behind or by getting into the anterior chamber, as in my third case. In addition to this, where the capsule is ruptured, the detritus of the cortex might block the outlets. The absorption of this detritus within the unruptured capsule, which Lange and others appear to believe in so firmly, seems to me highly improbable and entirely incapable of proof. A strong point in favor of the theory that the glaucoma is secondary is, that in my first two cases, the cataracts showed no signs of being in any way complicated by other disease when first seen, and that glaucoma has failed to appear in the other eye after a lapse of seven years in one case and three in the other. At any rate I feel like warning patients with one well-advanced cataract to report at more frequent intervals than I formerly did, and if the cataract shows signs of hypermaturity and can not be kept under observation, I believe it wise to operate on it even if the other

eye be good, provided that the patient has the prospect of living a number of years.

Of the authorities which I have consulted, some advise postponing the extraction in one eye so long as the other eye is fairly good. Others, without giving any good reason, advise operating on all ripe cataracts. The only suggestion that I have found of any real danger in postponing the extraction of a mature cataract is in Becker's contribution to the old Graefe-Saemisch, in which (Vol. VII, p. 269), after saying that an over-ripe cataract sometimes sinks down through breaking of the zonula by the contraction of the capsular cataract, (Becker considers this the result of a phakitis), he says: "The dislocated lens then lies as a foreign body in the vitreous and might (könnte eventuell) produce the same bad results as a couched lens." In other words he realized the possibility of such danger without apparently ever having known it to occur.

In closing, I wish to report another case, not strictly belonging in this paper, but which is interesting as an additional evidence of the danger of allowing Nature to take the entire charge of senile cataracts.

M. S., a German, about 57 years of age, presented himself with a cataract about one-half ripe, in the left eye and good vision in the other. I dismissed him with the usual advice to stay away until the sight of the good eye became involved. He returned after about eighteen months, with the lens entirely opaque, the cortex being much swollen and pressing the iris forward. The eye had no perception of light and had T.+1. He told me that he had not seen light for some time but did not think that involved any danger. The other eye was still good and remained so for many years thereafter. Both eyes had been moderately myopic from youth.

ON DILATATION OF THE PUPIL FROM STIMULATION OF THE CORTEX CEREBRI.*

By JOHN HERBERT PARSONS, M.B., B Sc.,
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BOCHEFONTAINE,[1] (1875) in describing various phenomena accompanying electrical stimulation of the brain of dogs, mentions dilatation of the pupil. Both dilatation and constriction were observed by Luciani and Tamburini[2] in their experiments. Ferrier[3] confirmed these results more in detail; thus, in monkeys: "(12) Including the posterior half or two-thirds of the superior and middle frontal convolutions, the eyes widely open, the pupils dilate and head and eyes turn to the opposite side." "(13) and (13[1]) On the anterior and posterior limb of the angular gyrus respectively the eyes move to the opposite side * * * usually also the pupils become contracted." "(14) On the superior temporo-sphenoidal convolution pricking on the opposite ear, head and eyes turn to the opposite side, pupils dilate widely." In dogs: "(7) On the frontal division of the second external convolution (coronal convolution) * * * the movements of the eyeballs I have found to be divergent, associated with contraction of the pupils." [I have not as yet been able to confirm this result.—J. H. P.] "(12) On the precrucial division of the sigmoid gyrus the eyes widely opened with dilatation of the pupils. * * * In one or two instances I have seen the eyeballs converge, but in all cases the pupils were dilated." [I have never yet obtained convergence accompanied with dilatation of the pupils.—J.H.P.] "(13) On the parietal and posterior division of the second external convolution movement of the eyeballs to the opposite side * * * the pupils occasionally contract."

Similar results were obtained by Ferrier upon jackals, equivocal results on rabbits. In cats he only occasionally obtained contraction, but does not state that he ever obtained dilatation. In pigeons the most constant effect of

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any was an intense contraction of the opposite pupil on stimulating the middle of the convexity of the hemisphere.

Horsley and Schäfer[4] obtained hemiplegia, accompanied by great dilatation of the right pupil and contraction of the left, in monkeys in which the anterior part of the right marginal convolution and almost the whole of the motor area had been removed.

Schäfer,[5] in exciting the visual center in monkeys, besides ocular movements, obtained movements of the eyelids, etc., which, he states, "have not been constant in my experiments, nor have the changes in the pupils which are sometimes observed accompanying the movements of the eyes. I have occasionally obtained from stimulation on or near the quadrate lobule marked contraction of the pupils. * * * More usually, however, the movements whatever the part stimulated, are accompanied by dilatation of the pupils."

Beevor and Horsley[6] state: "In the vast majority of instances there was no movement of the pupils; when it occurred dilatation was also noted. This dilatation of the pupils exists simply in association with turning of the head and eyes to the opposite side. It appeared to be represented most around the horizontal limb of the precentral sulcus, but we did not observe it many times."

Beevor and Horsley,[7] as also Franck and Pitres,[8] obtained dilatation of the pupils on stimulation of the forepart of the internal capsule.

Beevor and Horsley[9] in the orang-outang expressly state that there was no dilatation of the pupils from stimulating the brain anterior to the precentral sulcus.

Bechterew and Mislawski,[10] in investigating the centers for the secretion of tears, obtained also the usual effect of stimulation of the cervical sympathetic (dilatation of the pupil, action of the nictitating membrane, etc.), from excitation of the "inner parts of the anterior and posterior divisions of the sigmoid gyrus" in the dog. Whilst confirming these results I may incidentally remark that I have quite failed to obtain any conclusive effects upon the lacrymal gland.

My experiments have hitherto been confined to dogs and

cats, especially the latter, in which the movements of the iris are so readily demonstrated.

The animals were anæsthetised with A.C.E. mixture. A Du-Bois Reymond inductorium was used, with a single Daniel cell.

The following is a summary of the principal results obtained:

1. Dilatation of the pupil, like other effects of cortical excitation, is best obtained with slight anæsthesia. It often occurs without any movement either of the eyes or body, but most when the animal is struggling; it is never seen with deep anæsthesia.

2. It is only obtained from those parts of the brain which are concerned in eye movements—that is, from the anterior part corresponding to the frontal motor area for the eye muscles, and from the posterior part corresponding to the occipital "visual center." The frontal area in the dog and cat is in the neighborhood of the crucial sulcus, and the pupil effect is usually best obtained from the mesial surface of the hemisphere at this point. The occipital area is the posterior part of the second external convolution (Ferrier's).[3]

3. When well-marked, the dilatation of the pupil is accompanied by all the usual effects of stimulating the cervical sympathetic.

4. The effect is usually more marked upon the opposite eye.

5. The effect is diminished, but by no means abolished, by section of both sympathetic nerves in the neck. The other sympathetic effects are abolished.

6. The effect is abolished by section of the oculo-motor nerves intracranially, subsequent to section of the cervical sympathetic. Under these conditions the pupils assume the position of post-mortem equilibrium and are immobile. Hence, I conclude that in the absence of the sympathetic channels the diminished effect is due to inhibition of the tonic action of the third nerves.

7. The effect is not abolished on either side by the section of the corpus callosum. Stimulation of the cut surface of the corpus callosum results in the usual bilateral pupillary reaction.

8. Stimulation of the central end of a cut afferent or mixed nerve—for example, sciatic—still causes dilatation of both pupils after section of both cervical sympathetics—that is, a reflex inhibition of the third nerves.

9. I have not as yet obtained unequivocal contraction of the pupils from stimulation of the cortex, but not much attention has hitherto been paid to this point.

The following is the protocol of a typical experiment:

EXPERIMENT XIV.—JULY 14, 1900.—LARGE CAT.—A.C.E.

Sympathetics dissected out to the superior cervical ganglia. Left hemisphere exposed.

A.M.

11.00—Stimulate left sigmoid gyrus; eyes straight, botn pupils dilate.

11.02—Stimulate left second external gyrus; eyes to R., both pupils dilate. (The second external often gives a better result than the frontal, but did not in this case).

11.05—Left vagus and sympathetic torn out as high as possible.

11.10—Pupils contracted and equal.

11.11—Stimulate L. sigmoid; eyes straight; pupils equal and moderately dilated.

11.13—Pupils as at 11.10; stimulate L. second external; pupils as at 11.11.

11.15—R. vagus and sympathetic stimulated together; R. pupil dilates fully. R. vagus and sympathetic pulled out as high up as possible.

11.17—Pupils equal and moderately contracted.

11.18—Cat stopped breathing. Pupils equal and moderately dilated. Artificial respiration. Recovery.

11.23—Pupils very contracted. L. very slightly > R.

11.25—Stimulate L. sigmoid. Pupils slightly, but very sluggishly, equal; no eye movements.

11.30—Pupils react very feebly to light. 11.25 effect repeated twice.

11.40—Corpus callosum divided. Stimulate L. sigmoid; very sligh effect both. Stimulate L. second external; very slight effect both.

11.45—L. sciatic exposed and divided. Central end stimulated. Both pupils dilate well.

11.50—Stimulate L. sigmoid; no effect.

11.55—Stimulate L. sigmoid; well-marked dilatation of both pupils; no eye movements; no struggles.

NOON.—Deeper median section to insure division of the corpus callosum in its whole extent. Pupils moderatcly dilated.

P.M.

12.05—Stimulate L. sigmoid; no effect. The sigmoid gyrus has been considerably injured by the previous manipulations.

Stimulate outer part of L. sigmoid; marked dilatation of both pupils; struggles. Anæsthetic increased.

12.10—Repeat. L. eye down; no movement of right eye; dilatation of both, more marked on left side.

12.12—Stimulate L. second external; same as 12.10 effect.

12.15—L. hemisphere removed.

12.20—R. pupil widely dilated; L. pupil tightly contracted.

11.22—L. third divided intracranially. Both pupils moderately dilated and equal.

In the explanation of the results my remarks must be regarded as tentative. I believe that the pupillary reaction is entirely a reflex one, but is especially interesting as further demonstrating the possibility of a reflex inhibition. I can only in a very modified sense agree with Bechterew and Mislawski[10] that in the inner part of the sigmoid gyrus we have the central prolongation (*Verlängerungen, Fortsetzungen*) of the cervical sympathetic. It would be a misuse of the term to regard these results as pointing to the existence of cortical pupillary "centers."

BIBLIOGRAPHY.

[1] Bochefontaine. Société de Biologie, 1875 and 1876.

[2] Luciani and Tamburini. "Sui Centri Psico-motori Corticali," 1878 and 1879.

[3] Ferrier. "The Functions of the Brain," second edition, 1886.

[4] Horsley and Schaefer. Case 15. "Phil. Trans.," vol. clxxix B, p. 29.

[5] Schaefer. "Brain," vol. xi, April, 1898.

[6] Beevor and Horsley. "Phil. Trans.," vol. clxxix B, p. 213.

[7] Beevor and Horsley. Ibid., vol. clxxxi B, p. 82.

[8] Franck and Pitres. "Lecons sur les Functions Motrices du Cerveau," par Franck. Paris, 1387.

[9] Beevor and Horsley. Op. cit., p. 134.

[10] Bechterew and Mislawski. Ueber die Innervation und die Hirncentren der Thraenenabsonderung. "Neurol. Centralblatt," X, 16, S. 481.

POSTNEURITIC ATROPHY OF BOTH OPTIC NERVES DUE TO MUMPS.*

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[Translated by Adolf Alt, M.D.]

ON FEBRUARY 14, 1900, A. B., 25 years of age, came to me for consultation. He complained of a gradual loss of vision, dating from April of the previous year. With the left eye he could count fingers only at one meter, and with the right only at one-quarter meter. He, furthermore, had a very considerably limited visual field, yet he recognized colors fairly well. I found an atrophy of both optic nerves of that form which is so characteristic of a former optic neuritis.

The most careful anamnestic researches brought to light no other possible cause for this atrophy than an attack of mumps which he had in April, 1898, during his military service and for which he was confined to the hospital at Briançon for twenty seven days. Formerly he must have been a fine shot as he had received the decoration of the hunting horn which, in the Alpine regiments, the good marksmen wear on their sleeves. In December of the same year he had photopsiæ and when shooting he could not see as well as before. From April to May, 1899, his vision grew rapidly less. The chief physician of the Briançon hospital who examined him June 18 and 19, made even then the diagnosis of atrophy of the optic nerves. On June 20 he was sent to the fort, and on September 20 he was discharged.

Since his first visit to me he was subjected to a treatment consisting of the application of a constant current of five

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milliampères, and of injections of five milligrams of strychnia. Vision improved perceptibly, since he was enabled to count fingers at two and a half meters with the left and at one meter with the right eye. It was, however, impossible to gain more and this condition has remained the same since.

I had at first thought that this observation was unique, but when I searched the bibliography I found that this affection was well known to the military surgeons. I shall first cite an article from the *Memoires de Médicine et Chirurgie Militaires*, 1876, p. 305.

Considerations of visual disturbances with alterations of the papilla and the peripapillary zone observed during an epidemic of mumps in 1875–76, by Hatry (Surgeon-Major of First-class).

"A soldier of the Twelfth Cuirassiers came to the hospital on account of mumps of both parotid glands. He complained of visual disturbances, stated that he saw as if through a mist and that the white cornet of the sister of charity to him appeared blue.

An ophthalmoscopic examination revealed pathological changes which we have since seen in nine other patients suffering from mumps.

Three different degrees of visual disturbance are observed:

1. Vision is but little diminished. The papilla is not congested, there is no peripapillary infiltration; veins and arteries are full, no venous pulsation.

2. Visual acuity reduced, papilla hyperæmic, veins and arteries are overfilled, delicate circle of peripapillary infiltration.

3. Papilla much congested, can hardly be distinguished from the choroid; venous pulsation; large circle of infiltration, covering the papilla and the origin of the blood-vessels; visual acuity very much reduced; sometimes the color-perception is altered; smoke and mists; eyeball congested, upper lids swollen.

Interpretation: We have probably to deal with an afflux of blood which compresses the blood-vessels in the region of the parotid glands and secondarily cause intracranial and intraocular congestions. The prognosis is benign. Usually

the visual accuity returns to the norm in about two, or at most, three weeks.

In this soldier of the Twelfth Cuirassiers the affection is of the third degree. The papilla is of an uniformly bright red, especially in the outer segment; a ring of peripapillary infiltration twice the size of the papilla; identical changes in both eyes. V.=$^1/_X$. A few days later we saw whitish streaks along the veins and arteries in the retina. Fifty days after the onset the ophthalmoscopic picture had returned to the norm. V.=$^4/_V$."

Now follow nine almost identical observations made in the service of Major Hatry (Hôpital Desgenettes) by the Adjutant-Major Hocquard, and in the service of the Chief-Physician Alix by the Adjutant-Major S.

In all of these cases a congestion of the papilla, especially of its outer side, with peripapillary infiltration is found, and vision returned to the norm.

Finally, I want to relate an observation by Talon (1883, p. 103) an extract of which is in Mr. A. Blanchard's article (*Bulletin Méd.*, December 6, 1899), who himself has examined a further case of atrophy of the papilla after mumps.

Ocular troubles have very seldom been seen as complications of mumps. Hatry (*Arch. Méd. Milit.*, 1876, p. 303) was the first to draw attention to this subject, having observed a sufficiently large number of cases during an epidemic of mumps which occured at Lyon in 1875.

His observations may be divided into three groups and may be described in the following manner: (*a*) Diminished visual acuity combined with conjunctivitis and hyperæmia of the papilla; (*b*) Considerable infiltration of the papilla; (*c*) Visual acuity very much reduced, dischromatopsia, stillicidium, photophobia, vertigo. All of these symptoms were trancient and left no bad consequences behind.

This was not the same with the patient observed by Talon in 1883 (*Arch. Méd. Milit.*, 1883, p. 303). A young soldier had always enjoyed excellent vision. There was neither a hereditary nor acquired taint. About one month after he had been attacked by mumps very pronounced visual disturbances made their appearance in the right eye without apparent cause. The ophthalmic examination re-

vealed an optic neuro-retinitis. Two months later vision in this eye was lost by atrophy of the papilla.

The scarcity of these observations prompts us to publish a case of our own observation. This is one of atrophy of both papillæ and a trancient disturbance in the function of the right internal rectus, both coming on after an attack of mumps.

X, 23 years of age; father unknown; mother died probably of tuberculosis; no personal antecedents. Enlisted in 1898, was sent to the infirmary a few weeks later on account of mumps, which was epidemic in his regiment. There were severe headache, gastric disturbances, vomiting, vertigo and fever. After this rather violent onset, the disease had run its natural course, when, at the end of one month the patient noticed an almost sudden diminution of vision; objects appeared distorted and barely visible, being surrounded by a thick mist. Five or six days later he could only walk about with the greatest difficulty.

He was sent to the hospital and the following treatment inaugurated: Leeches were applied to the mastoid apophyses, ice on the eyes, and iodide of potassium internally. After a short time he was sent home to his family, as convalescent, led by a nurse, because he was utterly unable to guide himself.

Three months later we saw him for the first time and found: Slight bilateral exophthalmus, divergent strabismus of the right eye, the movements of which are very much limited toward the nose. There is then a paresis of this rectus internus. Both eyes are tearing and the congested conjunctivæ form chemosed elevations around the corneæ; the latter are normal. Both pupils are slightly dilated and react to neither accommodation nor light. The crystalline lens and vitreous body are both transparent.

The tension of the eyes is not increased, but when pressing them backwards the exophthalmus is momentarily reduced and a slight peculiar noise is heard, as if the eyeballs were rubbed against a serous bursa. No bruit on auscultation, no pain. Vision is very poor, the patient can not count fingers, and even when the hand is held very close to the eyes he only has the vague sensation of a shadow.

In the inverted image a discolored papilla is seen, the limits of which can only with difficulty be distinguished from the neighboring retina on account of a peculiar exudation which lies here and which we then considered to be the last remnant of a neuroretinitis. There is a striking contrast between the engorged veins and the very thin arteries; around the latter lie some patches of exudation.

We prescribed iodide of potassium, mercurial inunctions and tonic treatment. Under this management the inflammatory symptoms of the eyeball diminished; the conjunctiva took on its normal color, soon the chemosis and exophthalmus disappeared. The right eye is diverging slightly, but the movements toward the nose are much better, that is, the internal rectus is resuming its function.

Unfortunately, the same can not be said of the optic nerve. The papilla grows more and more white, it is clear that atrophy takes place; its borders are now more sharply defined, but white lines lie along the arteries as an evident sign of former inflammation.

It seems as if the visual function must be definitely lost; only light-perception remains, but no perception of objects. The patient can not guide himself. The same treatment is continued.

One month later, no divergent strabismus, not a trace of exophthalmus; pupils slightly dilated, reacting very feebly to light and accommodation; papilla white, vessels very thin. A hand placed very close to the eyes is perceived, but he can not count fingers. On a clear day he can guide himself without help. To sum up—his condition is somewhat improved.

Two months later we again saw the patient. He can now read the largest test letters at about one meter; the visual fields are very small—five centimeters in- and outwards, three up- and two and a half downwards for the left eye; the right field is a little larger. Dyschromatopsia. Movements good. The pupils react feebly to light and accommodation. Papillæ very white, blood-vessels very thin. Further and repeated tests of the visual acuity could not elicit an appreciable change.

To sum up—there was trancient paralysis of the internal

rectus, loss of vision due to neuroretinitis which had led to white atrophy. The treatment applied was followed by some good results, when we might have thought that vision was forever lost. A few months after the onset of his malady, X had been pensioned off with the following diagnosis: "Atrophy of both optic nerves, consequent upon an attack of mumps during an epidemic of this disease. This atrophy has made rapid progress during a furlough for convalescence. The usual methods of treatment have been unsuccessful."

Could not this neuroretinitis have been attributed to a hereditary or acquired taint? We have found nothing in the antecedents of the family. The patient does not acknowledge having had syphilis and we did not find any marks of it; neither of alcoholism nor of any other intoxication. The examination of his urine, repeatedly made, showed neither albumen nor sugar. No intellectual disturbances; no disturbance of sensibility or motility. Nothing abnormal in the different sinus of the face. The general symptoms of the beginning of the disease (high temperature, violent headache, vertigo, insomnia and gastric disturbances) were not sufficient to admit of the diagnosis of meningitis.

Before any examination was made one might have thought of a hæmorrhage in the fundus of the eye, a detachment of the retina or an embolism of the central retinal artery. Such an affection would, however, have been seen with the ophthalmoscope.

Atrophy of the optic nerves due to mumps was then the diagnosis we were forced to make. The relation between cause and effect was evident, and we have been able to follow every inflammatory stage of the neuroretinitis to the period of white atrphy of the papillæ.

However, how is such a complication produced?

Hatry, who had seen visual troubles appear when the mumps was at its highest development, thought he could explain them by congestions in the intracranial and intraocular cavities, due to the afflux of blood which compressed the vessels of the neck in the region of the parotid gland.

Talon thought that the alterations of the papilla and the peripapillary zone were symptoms of cerebral complications.

There is one cause which, we think, must not be lost sight of, namely—the infection. Like all infectious diseases, mumps may lead trancient or lasting complications in the visual apparatus, according to the less or more virulent character of the secreted poison. A very grave infection may so affect the visual system that it is fatally impaired in its function.

Yet, in order to produce such a result an infectious disease needs time, so that it may impregnate the organism with its toxins.

At least, this seems to be a fact with the forms of neuritis consequent on variola;[1] total amaurosis with slight neuroretinitis appearing in the period of convalescence from measles;[2] atrophy of both papillæ following typhus abdominalis;[3] neuroretinitis following influenza[4] and diphtheria.[5]

The same was the case with Talon's patient and ours. The visual disturbance came on late and when, so to say, the general disease had reached its evolution, reminding one of the relatively distant complications of diphtheria.

In their article on "Mumps," in the *Treatise of Medicine and Therapeutics*, Messrs. A. Legroux and L. Hudelo state that "the complications of the visual apparatus are rare and benign." Our observations, as well as those of Hatry and Talon, are such that they must modify the second part of this proposition.

Further, in a "General Review on the Nervous Complications of Mumps," by Mr. Gallavardin,[6] we find the following: "Already in 1853 Bergeron has noted the presence of mists which extended over the visual field. Hatry reports ten cases of amblyopia, including such with a mist only and others with a decided loss of visual acuity. Fournier adds nineteen cases and gives the following description:

A.—More or less pronounced diminution of visual acuity; confusing sensations of mists and smoke. In the nineteen

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[1] Arch. f. Ophth., 1866–71.

[2] Von Graefe.

[3] Leber and Deutschmann.

[4] Lebeau, Ophth. Rec., October, 1891; Vignes, Annal d'Ocul., cxv, p. 274.

[5] Pouchut, Gaz. des Hôp. 1873, p. 312; Galezowski, Rec. d'Opht., 1881, p. 214.

[6] Gaz. des Hôp., December 7, 1898, No. 144, p. 1329.

cases he notes sixteen cases of light amblyopia in which vision was only misty, and three cases in which it was bad enough to make reading impossible.

B.—Dyschromatopsia of transient character; finally in two cases he notes hemeralopia.

All of these disturbances are transient and disappear completely in a few days. They appear only in cases of very severe mumps and are doubtless due to some disturbances in the circulation. Hatry and Fournier have seen the congestion of the papilla and the surrounding retinal zone with the ophthalmoscope. In some of Hatry's cases the veins were tortuous, there was venous pulsation and the papilla could hardly be distinguished from the choroid.

We have, furthermore, to mention a case of iritis, by Bartreff; a case of paralysis of the accommodation, by Boas,[7] in a girl, 7 years of age, three weeks after mumps; and an, until now, unique observation, by Talon,[8] of a case of atrophy of both optic papillæ and blindness three months after mumps. Vision was completely abolished and both papillæ were absolutely white. This was certainly caused by the action of the poison of mumps on both optic nerves.

Finally, in March of last year,[9] I found the following circular addressed to the military surgeons:

"The Committee of Hygiene asks the Military Surgeons who may have cases of mumps under their observation to pay especial attention to the ocular lesions which may occur either during this disease or at some later period. The surgeon, Talon, has in 1883 made known a case of complete blindness for which, scientifically, no other cause could be assigned. Some other cases, although but few, are found in different medical publications.

This Committee has seen an analogous case. These cases are still so rare and they are of such great interest, not only for science, but also for the eventual rights of the patient to claim a pension, that every such observation should be studied with the greatest care and reported to the Committee."

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[7] Boas, Klin. Monatsbl., August, 1886.
[8] Arch. de Méd. Milit., 1883, p. 103.
[9] Op. cit., No. 3, 1899.

MEDICAL SOCIETIES.

THE TREATMENT OF CHRONIC GLAUCOMA.

A Discussion Before the Ophthalmic Section of the British Medical Association, at Its Annual Meeting at Ipswich, July–August, 1900.

I.—Francis Richardson Cross, F.R.C.S.,

Ophthalmic Surgeon Bristol Royal Infimary; Surgeon Bristol Eye Hospital.

I may perhaps be allowed to take the opportunity which this Annual Meeting of the British Medical Association affords to draw attention to the fact that a large number of cases of inflammatory glaucoma are still mistaken for iritis or other forms of inflammatory eye diseases, and to insist on the necessity for fully considering in the diagnosis of such cases the possible presence of a glaucoma, more particularly before active treatment by the use of atropine and mydriatics is recommended. We no doubt are agreed that the only reasonable prospect of recovery or improvement in "inflammatory glaucoma" (hæmorrhagic glaucoma to be an exception) is to relieve the strangulation of the eyeball by means of surgical interference, and that even the use of myotics is merely of temporary value until the services of a competent operator can be obtained.

In "simple glaucoma" the danger lies in the insidious and very gradual progress of the complaint. No definite symptoms enforce themselves on the attention of the medical man and the patient himself scarcely recognizes in the early stages that his sight is becoming defective. When he has done so, an incompetent adviser may possibly have satisfied him with the opinion that he is suffering with incipient cataract, and that he must patiently await the development of the disease until a degree of blindness has been reached which justifies

extraction of the lens. Ophthalmic surgeons (though fully aware of the serious nature of chronic glancoma) are not unanimous as to what is best to be done for patients under the different phases of the disease, and it will be interesting to get the opinions and experiences of those present at this meeting upon this important subject.

For the purpose of this debate on "The Treatment of Chronic Glaucoma," which I am invited to open, I have examined such notes as I possess of hospital and private patients, but I find it impossible to draw from them deductions based on statistics; some have paid only one or two visits; in some the notes are brief and incomplete; one can only use them as records of facts upon which one's experience and opinions are based. Forty-seven cases of chronic non-inflammatory glaucoma from my private casebook give more definite information than a much larger number which have been noted in hospital practice.

Chronic glaucoma, in which the manifestations of increased intraocular pressure are indefinite, and their progress very gradual, may be associated with a distint increase in eyeball tension or with a degree that is imperceptible.

The disease is usually discussed under two headings:

1. Chronic inflammatory glaucoma—progressive, with occasional exacerbations of congestion, which may be very temporary or of longer duration.

2. Simple glaucoma.

There may be varieties that seem intermediate between the simple and inflammatory forms of the disease.

A case that has been clearly diagnosed as one of chronic inflammatory glaucoma requires immediate operation by sclerotomy or iridectomy as imperatively as does an inflammatory case with acute or subacute symptoms. It is in glaucoma simplex that the difficulties arise in regard both to accuracy of diagnosis and to treatment.

Many of the objective symptoms of glaucoma are absent. There is no pain nor inflammation, and no peculiar appearance of the eyeball. The scleral veins are little if at all enlarged, the pupil is normal or but slightly dilated or sluggish, the aqueous chamber is but slightly shallowed, and the eyeball tension may be scarcely raised. There may be no sub-

jective symptoms of defective eyesight, and central vision is often practically normal.

But a closer investigation with the help of the perimeter and of the ophthalmoscope will enable us to clearly recognize the real nature of the disease. Ophthalmoscopic examination may show the presence of a pulsating artery (noted in about one-fifth of my cases) or of a deep and typical cupping of the optic nerve, but the appearance may be less characteristic, some degree of anæmia or atrophy of the disc, with possibly slight pushing of the vessels toward its nasal side, and may leave us in doubt whether the case is essentially an insidious glaucoma or an optic atrophy. Even a shallow cupping over the whole papilla may be due to the degeneration of atrophy and irrespective of any increase in the intraocular tension. Moreover, we commonly find that common glaucoma, especially in its later stages, is complicated by atrophy. Thus of the 47 cases quoted 20 were associated with definite atrophy, and in 4 the presence of glaucoma was scarcely recognizable. There was no note made of atrophy in the remaining cases, but it may have been present in some degree.

The perimeter is of special value in helping to distinguish between the two diseases. Defect in the nasal portion of the field of vision, though it is occasionally associated with uncomplicated atrophy, is strong presumptive evidence that the case is a glaucoma. A trustworthy observer has recently stated that by a study of the charts taken in some sixty cases of chronic glaucoma he finds that the field may first become defective in other directions, but my experience, based upon the careful chart-taking of my assistants for several years, has satisfied me that diminished vision on the nasal side (on the upper or lower quadrant of the nasal side) is the most important test that we can apply to the early detection of chronic simple glaucoma, while the retention of vision in the temporal and infero-temporal portion of the field is equally characteristic as the disease progresses. In the later stages, where atrophic changes are well marked, the characteristic appearances of the glaucoma field are lost, and there is found to be a general concentric contraction around the

fixation point, but even where this is narrowed to within 5 or 10° the central vision is often well maintained.

The color sense is said to be usually retained in glaucoma until late in the disease; while it is early impaired in optic atrophy.

I may have detained you too long on these points, but it is essential to realize the close association between optic atrophy and simple glaucoma (which has been designated "optic nerve atrophy with excavation,") and to estimate accurately the relative importance of each of them in cases where both are present together before we can form an opinion as to the probable value of operative interference.

Progressive simple glaucoma will sooner or later be complicated by atrophy, and if operation is to be of service it should be done before the limitation of vision approaches dangerously near the center of the field. If the patient is nearly blind, the atrophic changes are not likely to be arrested by the relief of the slight amount of excessive eyeball tension present; on the other hand, if there is sight worth saving, the best chance of doing so is by relief of the glaucoma.

As bearing on the prognosis in a case of simple or chronic glaucoma if left to itself, I would remind the meeting how often in an acute or subacute glaucoma insidious symptoms of increased eyeball tension have been present for a longer or shorter period, prior to the inflammatory stage of the attack. In 92 of my cases of acute or subacte glaucoma, I have it noted of 50 that definite prodromata had been present for some time previously. The notes were not taken with any special attention of making this point, so that we may be assured that premonitory symptoms had occurred also in many of the others.

These prodromata are analogous to the symptoms of a simple glaucoma, in fact I find it difficult to distinguish between them, and just as an inflammatory glaucoma is preceded by prodromata, so may any case of simple glaucoma become suddenly blinded by an acute inflammatory attack, or may be converted into a chronic inflammatory glaucoma by occasional temporary congestions. Moreover, the serious nature of even the most insidious cases is evident from the

effect produced on them by the action of mydriatics, or even by such moderate dilatation of the pupil as is associated with anxiety or with mental or bodily fatigue.

The cases of increased eyeball tension which are eventually operated upon with acute symptoms or under unfavorable conditions, have nearly all passed through a less evident but no less real condition of glaucoma, which might have been easily and effectually operated on at an earlier stage. It is comparatively simple to do a good operation on an eye that is painless and not congested, much more difficult on one that is inflamed and strangulated.

But while on the one hand pain and inflammation must be relieved (and if necessary by operative interference) there appears to be no such urgency during the indefinite symptoms of incipient or simple glaucoma. Not everyone will face the risk of an operation upon an eye whose vision is practically perfect, and of which no definite complaint is made. However well executed, it may be followed by at least a temporary deterioration of sight, and the patient and his friends may think that more harm than good resulted from the interference. It is comparatively simple to temporize with myotics and medical treatment. But will these effect a cure, or what is really the proper course to pursue as a remedy against the disease?

A slight simple glaucoma may perhaps occasionally be cured by the use of myotics, under comfortable surroundings, with rest and avoidance of worry. I, last week, saw a lady 40 years of age in whom I could detect no symptoms whatever of a glaucoma tendency, even under prolonged dilatation by cocaine, who, until within the last month or so had been continually using for two years pilocarpin drops for "hardening of the eyeball," under advice of an oculist in whose diagnosis I should put absolute dependence. Personally I do not remember to have ever effected a cure by drugs of simple glaucoma where the symptoms were definite, though one has often had suspicious cases in which the symptoms have disappeared, and others where slight definite symptoms have continued in abeyance.

Mrs. E. F., 30 years of age, consulted me in June, 1889, There was hyperæsthesia of the retina, halo, fogs and slight

subjective symptoms of glaucoma—with deep physiological cups and the vessels pushed aside—central vision and fields perfect. She was ordered eserine grain one-sixth ad ℥j, which relieved the rainbows and discomfort. I saw her once or twice up to August, 1891, when she was in the same condition, using the eyedrops from time to time as symptoms seemed to suggest. I have just ascertained that Mrs. E. F. has had but little trouble with her eyes since she last saw me.

Mrs. T. H., 51 years of age, came in April, 1892, complaining that for three years past she has seen haloes in both eyes when looking at any lights—candle, fire, match. Fogs and mists often accompanied haloes. V. $=^{5}/_{5}$, hm. 50. Fields perfect; large full eye; anterior chamber shallow. Nerve not definitely cupped, but central vessels on it pushed a litte sideways; indistinct arterial pulsation. T. +?

This patient has been using pilocarpin drops ever since 1892, seeing me once a year. June, 1900: She still has V. $=^{6}/_{7}$. The fields are quite full; the haloes and fogs at times are troublesome, otherwise she has no marked discomfort, but undoubtedly has simple glaucoma. T. +? I have put the question of operation before her, but she declined it until she is distinctly more inconvenienced or until some development of the disease takes place.

Where, as in this case, the glaucoma remains in the incipient stage, it may be justifiable to continue the treatment by myotics until there is definite evidence that despite their use, symptoms of tension are increasing—but then something further must be attempted.

A retrospect of my cases conclusively shows that, sooner or later, progressive deterioration of sight is the rule in simple glaucomas under medical treatment alone. A number of patients pay each a single visit, at which we can infer from the symptoms present, and from the past history of the sights, how little is the chance of improvement unless operation is resorted to? In many cases I have wished to operate, but the patient has declined it; in others atrophic changes seemed too far advanced to promise any good prospect from such interference; while an unfavorable condition of the general health is urged against operation in a certain number. I append a few notes of cases, some of which have done fairly

well for a long time under the treatment with myotics, some indicate the close association of atrophy of the optic nerve with simple glaucoma. In one, eyeball tension seemed to be kept in abeyance by the progressive development of myopia.

CASE I. *Very Gradual Impairment for Six and a-Half Years.*—Mrs W. H., 60 years of age, came in February, 1894, complaining of a discomfort and "dryness" of the L. E. V. $=^{6}/_{6}$ L. $^{6}/_{9}$, says she sees worse towards the nose, and nasal field is reduced by 10°. Both nerves are pale and depressed, not fully cupped, but vessels pushed aside, and arteries pulsate indefinitely. The patient was delicate and much inconvenienced by a very large uterine fibroid. I ordered pilocarpin drops. In August, 1898, still general eye discomfort, particularly in the conjunctivæ—V. $=$R. $^{6}/_{7}$, L. $^{6}/_{18}$. White atrophied nerves in both eyes, vessels somewhat pushed aside; indefinite pulsation. T. $+$1. July, 1900. This patient says she still sees very comfortably, and has continued the use of the drops for the past six and a-half years. Theoretically, I ought to suggest operation, but the patient is quite satified, and I know that an operation would be declined, while the suggestion of its necessity might aggravate the glaucoma.

CASE II. *Gradual Loss of Central and Peripheral Vision under Treatment by Myotics for Twelve Years.*—Miss B., 70 years of age, came on April 20, 1888, complaining of misty sight and haloes. V. R. $=$L. $^{6}/_{15}$. White nerves, vessels pushed outwards, arteries narrow, slightly pulsating. T. $+$ 1. No evident congestion of sclera, nor any narrowing ing of anterior chamber. Eyeballs healthy; iris a good color. Diagnosis: Simple glaucoma with atrophy. Ordered eserine drops, 1 gr. ad ℥j. November 14, sight is better; no haloes nor pain.

April, 1888.		January, 1890. June, 1890.		March, 1893.
R. $^{6}/_{15}$	.....	Each—$1^{6}/_{12}$	.....	$^{6}/_{24}$ J. 1
L. $^{6}/_{15}$—$1^{6}/_{12}$	.....		.....	$^{6}/_{20}$ J. 2
October, 1894.		January, 1896.		May, 1898.
$^{6}/_{60}$	.....	$^{6}/_{0}$	.....	R. fingers
$^{6}/_{14}$	.....	$^{6}/_{26}$	.....	L.—$1^{6}/_{36}$

Iridectomy was suggested to Miss B., but declined, as she was deaf, suffered from headache, and did not feel at all qual to an operation. No doubt there was considerable

complication of atrophy in this case, but the glaucoma was quite definite, though no active symptoms or congestions exhibited themselves. She has used eserine or pilocarpin throughout the whole twelve years she has been under my observation.

CASE III. *Improvement with Myotics.*—Miss B., 65 years of age, simple glaucoma of the left eye with much impairment of vision, central and peripheral. Marked improvement during eight months' treatment by pilocarpin, particularly of central vision—namely, from $^{6}/_{18}$ to $^{6}/_{9}$, and also to a large degree of field. Right eye has no symptoms of defect excepting a blur on the nasal side of field.

CASE IV. *Improvements of Peripheral Sight.*—Rev. W. L., 76 years of age, came on April 6, 1899, complaining that his sight (particularly for reading) had been failing much for the past year. Slight incipient cataract; simple glaucoma, pallor of the nerves. The fields of vision contracted, particularly in the upper nasal fields. After treatment by pilocarpin, the peripheral sight improved, although central vision continued to fail from R. $^{6}/_{12}$ to $^{6}/_{20}$, L. $^{6}/_{9}$ to $^{6}/_{15}$; this probably on account of progress of cataract, while the glaucoma element had somewhat abated.

CASE V. *Atrophy with Slight High Tension.*—R. H. P. S., 61 years of age. Essentially atrophy of optic nerve with deep cupping of nerve and dipping of vessels on nasal side. T. + 1. No congestion; smokes more than moderately. Central color vision defective, worse than peripheral. In July, 1899, no glaucoma was suspected, spectacles gave R. $^{6}/_{9}$, 1 J.; L. $^{6}/_{20}$, 4 J. A deep white physiological cup noted. In August, 1900, vision reduced to R. $^{6}/_{24}$, 6 J.; L. $^{6}/_{60}$, 16 J.; this improved a little under pilocarpin and strychnine injections. Operations were judged to be useless.

CASE VI. *Atrophy with Doubtful High Tension.*—Rev. G., 60 years of age, came in March, 1895, complaining of failing vision in the left eye for the past two or three years. R. E. saw well 1 J. and with $-2.50 = -$ cyl. $^{6}/_{7}$. L. E. saw badly 14 J., $-4\,^{6}/_{9}$. Peripheral fields perfect for white; in the left narrowed for red, and a little more hazy on the nasal side for red. Left nerve not cupped. A small speck of choroidal atrophy. The case was diagnosed as monocular optic

atrophy. He continued to work with R. E. until January, 1898, when he returned complaining of R. E. The left was practically blind; right with spectacles $^{6}/_{8}$, but field concentrally narrowed. Both nerves were white and cupped, the vessels pushed aside and suggestive of simple glaucoma. There was no appreciable tension, but probably an abnormally weak lamina cribrosa allowed of cupping from a very slight degree of pressure, not definite enough to be detected by the finger. The condition got progressively worse, both in central vision and impairment of the field, but the evidence of glaucoma was never certain, although sections of the eye might probably have proved its presence by showing contact of the ligamentum iridis with the periphery of the cornea at the angle of the anterior chamber. In December, 1898, he could still read moderate print, but was getting worse.

Case VII illustrates the progress of a case of insidious chronic glaucoma under myotics for seven years, in which definite progressive development of a myopia seemed to have a beneficial effect as against a rise of intraocular tension. No alteration has been observed throughout in the cornea or aqueous chamber, nor has there ever been marked deterioration of the vitreous humor. Miss J., 51 years of age, came in May, 1893, complaining of loss of vision in R. E., which was reduced to letters of 15 J. L. E. 1 J. — 3.50 $^{6}/_{9}$. Nothing was noticed to account for the sudden failure. Tension was slightly high. Rather deep physiological cup. Pilocarpin ordered. In a month vision 6 J. — 3.50 $^{6}/_{12}$, and shortly afterwards 1 J. and — 3.50 $^{6}/_{9}$. Both eyes seemed to have a tendency to simple glaucoma, and pilocarpin was continued for both. This lady has been continuously under observation ever since; she has throughout had distinct symptoms of chronic glaucoma varying much in degree; at times well-marked pulsation of the central artery and slight definite rise in tension, but at other times these were absent, and no nerve cupping could be detected; there have never been pronounced symptoms nor congestion. In June, 1895, the myopia slightly increased in amount from 3.50 to 4.50 in L. E. In December, 1897, it was 6 D. and 10 D.; and in April, 1900, the vision was R. — 11 $^{6}/_{9}$, L. — 12 $^{6}/_{9}$, and 1 J. in each. The visual fields have continued in much the same condition for

the past four years. Possibly here I should have done better by operation, but reasons make it difficult to do more than suggest that something may have to be done.

CASE VIII.—Miss B., 41 years of age, came on May 20, 1889, complaining of right eye always uncomfortable with ophthalmic neuralgia, and, when tired, fogs and haloes. Vision R. $-1.50\ ^{6}/_{12}$, L. -1 cyl. $^{6}/_{6}$; R. field narrowed on temporal side. The next visit paid was on January 14, 1892, when both eyes had simple glaucoma; right eye worse, and field more contracted peripherally. January 22nd. Iridectomy was done on both eyes; this gave comfort, but the sight continued defective for several years, during which the patient was passing through the menopause. In March, 1898, she was very much better; the left eye practically perfect, the right field of vision still defective in the lower nasal portion. Though the fields of vision were defective, and even got worse for a time after the operations, the condition of the eyes was much more satisfactory ultimately. The patient has now perfect vision in the left eye, and practically perfect vision in the right. Without the operations her condition before now would have been very critical.

CASE IX.—Miss D., 67 years of age, seen in April, 1895, for left herpes frontalis, apparently not affecting the eyeball. Vision R. $+0.50\ ^{6}/_{7} + 3$ J. 1; L. $+0.50\ ^{6}/_{9} + 3$ J. 1 (?). Central guttate choroiditis in both. R. media clear, optic nerve normal. L. media hazy, optic nerve pale and cupped. T +?. In August eye was better, but was ordered pilocarpin. Miss D. next came in March, 1897; she had used pilocarpin every second day for four years. The right eye was good. The left eye V. $+^{6}/_{18}, +1\ ^{6}/_{12}, +3.5$. 4J. The field was narrowed. September, 27, 1897, field very much narrowed. V. $+1\ ^{6}/_{18}$. Iridectomy of left, with marked improvement of field and of central vision; but in October, 1898, the eye was less well and the field more contracted. I have not seen her since. The right eye was free from glaucoma.—*British Medical Journal.*

[TO BE CONCLUDED.]

ABSTRACTS FROM MEDICAL LITERATURE.

By W. A. SHOEMAKER, M.D.,

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RAINBOW VISION.

Adamkiewitz (*Neurologisches Centralblatt*, July 15, 1900) calls attention to a peculiar series of phenomena which he believes are the result of constriction of the central artery of the retina. It begins with a feeling of tension in the eye, and then a slight veiling of the field of vision. If now the subject looks at a luminous object it appears to be surrounded by rings which show the colors of the spectrum. The most common cause of this appears to be a cold douche after a sojourn in a steam bath. Occasionally it may be produced by heat, as for example, if a patient sits under a gas flame, in the winter, and the head becomes warm. It is particularly common at night. It seems to have no clinical significance, but is interesting as showing in certain cases that the sympathetic system may act upon a single small blood-vessel, and it constitutes the first definite proof hitherto offered of the effect of psychical excitement upon the caliber of the blood-vessels.

CONCERNING YELLOW AND YELLOWISH-GREEN EYEGLASSES.

V. N. Dolganoff and Klimovitch (*Vratch*, July 22, 1900) investigated the composition, physical properties and physiological effects of yellow and yellowish-green glass, and conclude that this form of glass may be employed with advantage in the following circumstances:

1. In all cases in which the eyes must be protected from an excess of violet or ultraviolet rays, as for example, in working under strong electric light—in such cases the preference must be given to yellowish-green glass.

2. Whenever the eye lacks the natural protecting ele-

ments, *e. g.*, in aphakia, in which, according to Widmark, the absence of the crystalline lens deprives the eye of the protection from an excess of violet rays, especially if the patient, for any reason, is suffering from weak eyesight.

3. An important property of these glasses is to facilitate distant vision, and this has already been recognized by the German military authorities. The eye armed with yellow glasses distinguishes objects at a greater distance than the naked eye, a fact which is of great importance in target practice.

These yellow eyeglasses may also be employed as protectives against an excess of violet and ultraviolet rays in working with very strong sources of light, *e. g.*, molten metals, etc.

CATARACT REMOVAL IN PERSONS WITH BUT ONE EYE.

Fritz Mendel (*Berliner Klinische Wochenschrift*, June 1, 1900) states that in the past eight years nine patients have come to Hirschberg's clinic who had lost an eye by an operation for cataract in the other eye. The unfortunate results of the first operation make the success of a second operation extremely important. A thorough aseptic preparation is the chief condition of success. The tear apparatus is a common source of infection, and if any inflammatory condition exists it must be thoroughly treated. The thorough antiseptic treatment of the nose for some time before the operation is also advised. In addition to this the lids must be thoroughly prepared. General anæsthesia permits of much more thorough asepsis, and is advised in these cases. Preliminary iridectomy is also advised, and the operation for cataract is undertaken three weeks later. All of the nine cases were operated upon with success.

THE TREATMENT OF KERATOCONUS WITH GALVANOCAUTERY.

Herman Knapp (*Journal of the American Medical Association*, August 18, 1900) gives the following rules as to the manner of application: Never cauterize too deeply, and if the result of the first operation is imperfect, apply the convex disc electrode again to the place in which you want the

subsequent cicatricial contraction to have the greatest effect. Spare, if in any way possible, at least half of the pupillary area. If we operate early, even in restricting, as we should, our interference to the progressive cases, we have to deal with a clear cornea the reaction will be least and the visual result as well as the operative effect, greatest. We should not operate when the least septic condition is present in the conjunctiva or in the lids, and especially in the lacrymal sac.

THE EFFECTS OF FLASHES OF ELECTRIC LIGHT ON THE EYE.

Dunbar Roy (*Medical News*, September 8, 1900) believes there have been more cases observed than reported of temporary injury to the eye through intense electric flashes. The danger of electric light is illustrated by a series of cases from practice. In a number of cases cessation from school work has had to be advised because of the injurious effect of incandescent lights; and in other cases student's lamps had to be substituted. Electric ophthalmia is discussed. It seems probable that lights are injurious to the eye in proportion to the amount of ultraviolet rays they contain. The Welsbach gas light seems less injurious than others. There should, however, always be an under shade, as well as upper, to modify the light.

RETINITIS ALBUMINURICA.

Francis W. Alter (*American Medical Compend*, July, 1900) draws the following conclusions:

1. Retinitis albuminurica indicates a high toxic condition of the blood which, in chronic kidney disease, will prove fatal in from one to two years.

2. The eye symptoms in such cases are always the first definite demonstrable lesions of chronic disease, particularly is this true of the shrunken kidney.

3. Retinitis from Bright's disease assumes two forms—exudative and degenerative. In the first, occurring in acute nephritis, recovery with serviceable vision is possible. In the second, connected with chronic nephritis, recovery is impossible.

4. Retinitis occurring in pregnancy gives evidence of a

toxic condition of the blood which endangers the life of both mother and child, and unless remedies prove efficacious, abortion is justifiable, especially when it occurs in the early stages of pregnancy, or if a previous pregnancy has already injured the retina or the optic nerve.

5. The appearance of the lesion in albuminuria, first described by Liebreich, does not agree with the ophthalmoscopic picture in the majority of cases.

The author concludes with a plea for the more general use of the ophthalmoscope by the general practitioner.

INFECTION THROUGH THE CONJUNCTIVAL SAC.

Mayer (*Münchener medicinische Wochenschrift*, August 21, 1900) made a large number of experiments on animals, demonstrating the possibility of bacterial invasion of the circulation, and generalized infection through the medium of the conjunctiva. This takes place with great rapidity after inoculation with chicken cholera, mouse typhus, anthrax, peste, in small animals with glanders and psittacosis (Nocard). Pseudo-tuberculosis gives a subacute course; in larger animals psittacosis, glanders, and tuberculosis are very chronic. Tetanus and diphtheria are fatal through the formation of toxins; diphtheria and the staphylococcus pyogenes aureus produce local infections, while the organisms of cholera, typhoid and actinomycosis were not able to invade the system; the tears appearing to have a special inhibitory effect on cultures of the former two, while, on the contrary, the growth of the diphtheria bacillus was encouraged.

VISION AND SEASICKNESS.

Arthur E. Sumne (*Ophthalmic Record*, July, 1900) believes that seasickness is an ocular phenomenon, and in evidence of this presents the case of a house that was carried away by a flood and deposited in a very abnormal position. Though perfectly stationary, it made persons who entered it seasick. The disturbed relations affected the field of vision in such a way as to produce the reflex symptoms of *mal de mer*. He believes the disturbance is really a psychic one, caused by complicated mental impressions which produce systemic disturbances.